Br Heart J 1984; 52: 688–9

Correspondence

Shoshin beriberi: a rare diagnostic problem

Sir,

The description by Engbers and colleagues of a case of profound circulatory collapse in an alcoholic (1984; 51: 581–2) is interesting as a description of recovery from severe overindulgence in alcohol, but the contention that the entire clinical presentation was due to thiamine deficiency is not supported by the biochemical data presented. The low erythrocyte transketolase activity should be ascribed to thiamine deficiency only if it is corrected by the addition of thiamine pyrophosphate in vitro. When this test was carried out the increase in transketolase activity was within the normal range. If the addition of thiamine pyrophosphate does not significantly increase the transketolase activity, it is difficult to sustain the argument that the patient is thiamine deficient.

As the authors state, depressed transketolase activity may occur in alcoholics with liver disease, and in patients with longstanding thiamine deficiency, but it has not been shown that the synthesis of transketolase is regulated by the availability of thiamine. Low transketolase activity in the presence of excess thiamine pyrophosphate may well be caused by abnormalities due to chronic alcoholism which are independent of thiamine deficiency. Our understanding of the relation between thiamine deficiency and heart failure will be improved only when more direct assays (for example, the thiochrome method[1]) are in routine use.

Mark Dancy,
M K Gaitonde,
J D Maxwell,
St George's Hospital,
London SW17 0QT.

Reference

1 Dancy M, Evans G, Gaitonde, MK, Maxwell JD. Blood thiamine and thiamine phosphate ester concentrations in alcoholic and non-alcoholic liver diseases. *Br Med J* 1984; 289: 79–82.

This letter was shown to Dr Engbers and colleagues, who reply as follows:

Sir,

We agree with Dr Dancy and colleagues that thiamine deficiency in our patient is not proved biochemically by a high thiamine pyrophosphate effect. As they state, the low transketolase activity in chronic alcoholism may be independent of thiamine deficiency. The thiamine pyrophosphate effect is clearly not a sensitive test to diagnose thiamine deficiency, which calls for a more direct assay.

In spite, however, of the lack of direct biochemical evidence of thiamine deficiency it can safely be concluded from the clinical picture of our patient that overindulgence in alcohol cannot be its only cause. This is because of the high cardiac output with a predominantly right sided heart failure and the rapid recovery after treatment with thiamine alone, while he had already ceased to take any alcohol two weeks before admission.

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G P Molhoek,
A C Arntzenius
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The Netherlands.

Prevalence of coronary artery disease in patients with isolated aortic valve stenosis

Sir,

In their recent paper (1984; 51: 121) Exadactylos *et al* refer to my paper on "Angina, coronary disease, and aortic stenosis" as having been published only in abstract form but never later in full. In fact, the study was published in full in the *American Heart Journal* (1977; 93: 382). This paper has been cited repeatedly by authors on this subject, including Thompson *et al* in the *British Heart Journal* (1979; 42: 447).

With reference to one of the issues discussed, our experience since 1977 has confirmed that significant coronary artery disease can be present in patients with aortic stenosis without angina.

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